Rhizomania is considered the most serious disease of sugarbeets worldwide because it causes severe loss, is difficult to control, and persists in the soil almost indefinitely once established. It is caused by beet necrotic yellow vein virus (BNYVV) and vectored by the parasitic soilborne fungus Polymyxa betae. The virus is incapable of infecting roots without being introduced by zoospores of the vector. Rhizomania was first reported in northern Italy in 1952 and has since spread to nearly all areas of the world where sugarbeets are grown. Although the disease will only move a few inches per year through soil without aid, it is easily spread by infected plant material, within soil adhering to non-host root crops, agricultural equipment carrying contaminated soil, irrigation water, wind, and any other means that can move even small amounts of soil. It is not, however, seed transmitted.

Rhizomania has now found its way into all sugarbeet growing areas in the United States. Originally identified in 1982 in the western hemisphere in Paso Robles, California, it had been thought that the disease would not become an important problem in the cooler, northern tier of states where sugarbeets are grown, but experience has made it clear that this is not the case. In Idaho, rhizomania was detected in only 27 fields (670 acres) out of 354 fields sampled (14,740 acres) in 1992, the first year it was diagnosed. Since that time the disease has spread considerably. Many growers have experienced severe economic loss, and several have had complete loss. In 2004 we now estimate that at least 40%, or more than 90,000 acres, are affected in the Amalgamated Sugar Company growing area of Idaho, eastern Oregon, and Washington. This is a dramatic increase from its first detection and from 10%, or 23,000 acres that were estimated in Idaho in 2000.

In Montana, rhizomania was confirmed in both 2003 and 2004. Our experience in other areas strongly suggests that it has probably been in Montana for several years. After the disease is introduced into a field, usually two or three sugarbeet crops are required before symptoms are evident. Although there is no specific data, estimates are that between 10% and 30% of the acres in Montana have some level of infestation.

**SYMPTOMS**

Roots are stunted and there is a proliferation of lateral rootlets, giving the root a bearded appearance. The taproot may be constricted and there may be excessive crown growth, giving the root a wineglass shape. Vascular tissue is discolored and the taproot may be rotted. With mild infections, there may only be slight lateral rootlet proliferation on the taproot or lateral roots. The vascular discoloration may not be very obvious in mild infections, but in longitudinal section the vascular bundles have a disorganized or “marbling” appearance in the
vicinity of rootlet proliferation. This is in contrast to the parallel vascular bundles in normal, healthy tissue.

Leaf symptoms consist of slight yellowing, erect growth habit, and leaf proliferation. Leaves are usually uniformly chlorotic. In mild infections, there may be little or no stunting of the foliage, normal growth habit, and a slightly perceptible but uniform lime-green leaf color. This contrasts with nitrogen deficiency where older leaves are often chlorotic while younger leaves are green. Plants often wilt during the warm part of the day. Yellowing of the veins is a symptom of systemic infection but is rarely seen.

**EPIDEMIOLOGY**

Both the virus and fungal vector are obligate parasites in that they require a living host to reproduce. Both are host specific and the virus is dependent on the vector for infection. The natural host range, therefore, is limited by those species that *P. betae* is capable of infecting, and includes primarily species in the same genus as sugarbeets (*Beta* sp.), spinach (*Spinacia oleracea*), and a few species in the genus *Chenopodium*. Experimentally the virus can be mechanically transmitted to several species in the families Chenopodiaceae, Aizoaceae, and Amaranthaceae.

The vector, *Polymyxa betae*, indefinitely survives in the soil as cysts or groups of cysts called cystosori. Disease is favored by high soil moisture, warm soil temperature (optimum 77°F), short rotation, and neutral to alkaline soils. With free water and soil temperature of about 60°F and above, individual cysts germinate in the vicinity of sugarbeet roots and release a single zoospore that infects primary root tissue, carrying the virus into the plant cell. Severity of infection is directly related to the population of viruliferous *Polymyxa* in the soil. A plasmodium develops within each infected cell which then either develops into a zoosporangium or a cystosorus. The zoosporangium releases secondary zoospores to the exterior of the root that then produce additional infections. The process continues until the rootlet dies and the plant produces new rootlets, which then support continued infections. Many infection cycles can occur during a single growing season.

The plasmodium may also develop into a cystosorus. It’s unclear what stimulates the change from zoospore to cystosorus production, but cystosori are groups of mature resting spores that have been shown to survive for as long as 20 years in air-dried soils. A single cystosorus can contain as many as 300 individual cysts within a single plant root cell.

**DISEASE MANAGEMENT**

**Resistance:** The most effective method to control rhizomania is to plant resistant cultivars. The resistance is partial and is based on a single dominant gene. A resistant cultivar should be planted if there has been any field history of rhizomania, no matter how small the affected area. If rhizomania has been found in another field on the farm, or in a nearby field, the likelihood that most or all fields are already infested is very high. Planting resistant cultivars will make long-term disease management far more effective. At least two or three sugarbeet crops are necessary after a field is first contaminated with rhizomania before noticeable
symptoms develop. With such a long incubation period, once we observe and diagnose the disease, the inoculum has likely already increased to a high level.

Available cultivars are not immune to rhizomania, but the partial resistance is sufficient to achieve good yields when managed properly. In 1999 tests under severe disease field conditions the best cultivars yielded 22.35 tons/acre with 15.35% sugar. In comparison, the standard susceptible check cultivars yielded 9.30 tons/acre and 13.80% sugar. Under mild rhizomania conditions in 2003, the best cultivars yielded 35.07 tons/acre with 15.92% sugar, while the susceptible check cultivar yielded 25.53 tons/acre with 13.45% sugar. The Idaho-Oregon-Washington growing areas require the highest curly top resistance of any other area in the U.S. Initially rhizomania resistant cultivars were very low in curly top resistance, but this continues to improve as new cultivars are developed. Rhizomania resistant cultivars now have nearly the same curly top resistance as standard cultivars.

**Rotation:** Planting resistant cultivars alone will not allow maximum yields to be achieved. A minimum 4-year rotation is essential for good management. For example, our research has clearly shown that with the best resistant cultivars and best management practices except for rotation, longer rotations result in increased yield. From 1995 through 1999 at the University of Idaho, we measured 16.1 tons with back-to-back beets, 18.7 tons with a 2-year rotation, 23.8 tons with a 3-year rotation, and 30.7 tons with a 4-year rotation. Short rotations are a poor decision for management of any disease, and can be disastrous with rhizomania because high populations of the pathogen develop and last in the soil for many years. Reduction of the pathogen population with rotation to non-host crops does not occur as rapidly with rhizomania as with most other soilborne diseases. Data from preliminary experiments indicate a benefit from planting oilseed radish and incorporating as a green manure after eight weeks growth.

**Soil Moisture:** Sugarbeets grow best when soil moisture is kept between –40 and –60 centibars (cb). Obviously it’s not possible to irrigate and continually maintain these levels of soil moisture, but excessive irrigation should be avoided. When soil is wetter than –40 cb (i.e., –30 to 0 cb), infection by *Polymyxa* increases greatly. After planting and prior to seed germination, when soil temperatures are lower than required for infection, it is best to fill the soil profile then wait as long as possible before applying additional irrigation. The disease will be more severe on fields with compacted soil, in low spots, areas with poor soil structure and inadequate drainage, and other places where soil moisture is excessive.

**Site Selection:** Know the field history before planting. Whenever possible, choose fields without a history of rhizomania. If that’s not possible, choose a site where the recommended 4-year rotation can be maintained.

**Early Planting:** When young plants become infected, loss from rhizomania will be greatest. The disease becomes active with soil temperatures between 50° F and 59° F, and early planting to establish the crop before infection occurs will reduce losses.

**Stand:** Good plant populations can help reduce the severity of rhizomania. Closing the rows early with high plant populations tends to maintain cooler soil temperature, which can reduce the rate of disease development.
A NEW STRAIN (PATHOTYPE) OF THE RHIZOMANIA VIRUS AND ITS POSSIBLE IMPACT ON THE SUGARBEET INDUSTRY

In the summer of 2002 and 2003 several fields with BNYVV-resistant cultivars in the Imperial Valley of California, were observed with severe rhizomania symptoms. Standard soil baiting with sugarbeet seedlings followed by enzyme-linked immunosorbent assay (ELISA) were conducted. Resistant cultivars grown in regular BNYVV-infested soil remained resistant, but when grown in Imperial Valley BNYVV-infested soil, all resistant varieties tested susceptible based on elevated ELISA values. These results indicated that rhizomania resistance had been compromised by a new strain of the virus in the Imperial Valley. This work was conducted by Dr. Hsing-Yeh Liu, USDA-ARS, Salinas, California.

Three strains of BNYVV have been reported worldwide. Strain A is distributed through most sugarbeet growing countries, including the U.S. Strain B has only been reported in Germany and France. Strain P is more aggressive than the A and B types and can infect partially resistant cultivars. It has only been identified in a region around the French town of Pithiviers, and in East Anglia in the United Kingdom. Through his research, Dr. Liu has determined that the Imperial Valley strain (IV-BNYVV) has likely evolved from the existing A strain.

Probable New Strain(s) in Idaho and Minnesota: In 2004, soil-baiting bioassays were conducted on soils from Idaho and from the Red River Valley of Minnesota. In preliminary tests, one soil sample from Idaho and five of six soils from Minnesota were found to contain resistance-breaking strains of BNYVV. Differential response of four cultivar seedlings suggest that there are three strains of BNYVV in the soils from Minnesota. All fields where new strains have been identified have had a history of heavy cropping to sugarbeets with short rotations. These results support the hypothesis that these strains are a small part of the variable virus population and are selected with intense cropping of resistant cultivars. Confirmation tests on these soils are currently being conducted in California and Idaho.

The emergence of resistance-breaking virus strains is due to genomic variation, primarily mutation. However, most mutants are either not viable or not positively selected. The widespread planting of resistant cultivars likely imposes positive selection pressure for mutants that have the ability to overcome existing resistance, which leads to partial or total breakdown of the resistance. The durability of beet cultivars that are resistant to BNYVV is currently being assessed, and additional sources of resistance with different genetic determinants are being sought to increase the stability and durability of the resistance.

Disease and Resistance History: Rhizomania was first diagnosed in the Imperial Valley of California in 1990, and the new strain was found in 2002, 12 years later. The disease was first found in south-central Idaho near Rupert in 1992. In 1996, it was identified in the Treasure Valley of southwestern Idaho near Middleton. It has been 12 years since the disease was first diagnosed in the Magic Valley and 8 years since the disease was first diagnosed in the Treasure Valley. Since 1996 Idaho growers have been planting rhizomania resistant cultivars in known infested fields.
Short rotations increase selection pressure on the pathogen and reduce the time before new strains become dominant in the population. Far too many growers are continuing to plant sugarbeets on one- and two-year rotations. In rhizomania-infested fields, a three-year rotation is common, but a four-year rotation is strongly recommended. With a four-year rotation, it will obviously take four times as long for the selection process to occur.

The current situation is considerably different than it was in 1992. At that time, resistance to BNYVV had been discovered nine years before, and rhizomania-resistant cultivars were already being planted in California. At this time, there has not yet been resistance identified to the new pathogenic strain of BNYVV.

**CONCLUSION**

Growers in Montana and Wyoming have the opportunity to benefit from the experience we’ve had with the disease in Idaho. When a grower has the disease diagnosed on one field, all fields should be planted to a resistant cultivar. Shortened rotations and/or planting susceptible cultivars will greatly exacerbate the situation and result in larger losses from this disease in the future. Planting to a susceptible cultivar will greatly increase disease inoculum and result in yield loss when the partially resistant cultivars are subsequently planted. Shortened rotations likely will result in selection of resistance-breaking strains of the virus before adequate resistance to these strains is available commercially. Lengthening the sugarbeet rotation is the best way to gain time for the identification of resistance, breeding new resistant cultivars, and developing other rhizomania management practices.

There are examples that complacency and ignoring the disease are resulting in severe losses. It is unfortunate that we frequently need to experience major problems to be reminded that the threat is by no means idle. To manage rhizomania, it is essential to sample suspected fields and correctly diagnose the problem. There is no reason to disregard or overlook rhizomania. We have the tools for good management, and we can take the steps necessary to achieve acceptable yields.